The effects of sleep restriction and altered sleep timing on energy intake and activity energy expenditure

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McNeil J, Doucet É, Brunet J-F, Jaeger Hintze L, Chaumont I, Langlois É, Mailtland R, Riopel A, Forest G. The effects of sleep restriction and altered sleep timing on energy intake and activity energy expenditure. Physiology & Behavior, 2016, 164: 157-163. https://doi.org/10.1016/j.physbeh.2016.05.051

Abstract:

Experimental evidence suggests that sleep restriction increases energy intake (EI) and may alter energy expenditure (EE). However, it is unknown whether the timing of a sleep restriction period impacts EI and EE the following day. Hence, we examined the effects of sleep restriction with an advanced wake-time or delayed bedtime on next day EI and EE. Twelve men and 6 women (age: 23 ± 4 years, body fat: 18.8 ± 10.1%) participated in 3 randomized crossover sessions: control (habitual bed- and wake-times), 50% sleep restriction with an advanced wake-time and 50% sleep restriction with a delayed bedtime. Outcome variables included sleep architecture (polysomnography), EI (food menu), total EE and activity times (accelerometry). Carbohydrate intake was greater on day 2 in the delayed bedtime *vs.* control session (1386 ± 513 *vs.* 1579 ± 571 kcal; $P = 0.03$). Relative moderate-intensity physical activity (PA) time was greater in the delayed bedtime session *vs.* control and advanced wake-time sessions on day 1 (26.6 ± 19.9 *vs.* 16.1 ± 10.6 and 17.5 ± 11.8%; $P = 0.01$), whereas vigorous-intensity PA time was greater following advanced wake-time *vs.* delayed bedtime on day 1 (2.7 ± 3.0 *vs.* 1.3 ± 2.4%; $P = 0.004$). Greater stage 1 sleep ($\beta = 110$ kcal, 95% CI for $\beta = 42$ to 177 kcal; $P = 0.004$), and a trend for lower REM sleep ($\beta = -20$ kcal, 95% CI for $\beta = -41$ to 2 kcal; $P = 0.07$), durations were associated with greater EI between sleep restriction sessions. These findings suggest that the timing of a sleep restriction period impacts energy balance parameters. Additional studies are needed to corroborate these findings, given the increasing prevalence of shift workers and incidences of sleep disorders and voluntary sleep restriction.

Keywords: Food intake | Physical activity | Sleep architecture | Bedtime | Wake-time

Article:

1. Introduction

Borbély [1] suggested that sleep is regulated by 2 overlapping processes: the homeostatic sleep drive (or process "S") and the circadian rhythm (or process "C"). The homeostatic sleep drive (process "S") promotes the occurrence of slow-wave sleep (SWS) as the amount of this sleep stage is greatly influenced by the length of prior wakefulness [2]. Conversely, REM sleep is mainly influenced by the circadian rhythm (process "C") and is more common during the second part of the night when core temperature is reduced and hypothalamic-pituitary-adrenal (HPA) axis activity and cortisol release are greater [3]. Sleep restriction protocols [4], [5] comparing sleep architecture when anchoring the sleep period during the first or second half of the night reported no differences in SWS between sleep restriction protocols, whereas REM sleep was greater during sleep held the second half of the night. Stage 2 sleep duration was consequently reduced as a result of maintained SWS and greater REM sleep durations during this time.

Studies have reported mean increases of ≈ 300–500 kcal over 24 h following an imposed sleep restriction condition *vs.* a control condition (habitual sleep duration) [6], [7], [8], [9], [10], [11]. However, the effects of imposed sleep restriction on energy expenditure (EE) are not as consistent; some studies reported no changes in EE inside a lab/inpatient clinic [8], [12] and under free-living conditions [11], whereas others reported either greater [7] or lower [13] EE following similar sleep restriction protocols (1–2 nights of 4 h in bed/night). Studies have also reported negative associations between SWS duration and energy intake (EI) the following day under habitual sleep conditions [14], as well as negative associations between changes in SWS and REM sleep with changes in carbohydrate and fat intakes between a habitual and partial sleep restriction condition [15]. Finally, Gonnissen et al. [16] reported greater post-dinner desire to eat ratings following 1 night of fragmented sleep, which caused a significant reduction in REM, but not SWS time, compared to 1 night of non-fragmented sleep (control condition).

Taken together, these studies suggest that reduced sleep duration increases EI and may affect EE. However, it is unknown whether imposed alterations in sleep timing, in addition to reduced sleep duration, have an effect on EI and EE the following day. The primary objective of the present study was to evaluate the effects of a 50% sleep restriction with an advanced wake-time or delayed bedtime on EI and EE over 36 h. The secondary objective was to assess the strength of the associations between changes in sleep architecture with changes in next day EI and EE between sessions. It was hypothesized that sleep restriction with an advanced wake-time would lead to greater EI coupled with lower EE and moderate-to-vigorous physical activity (PA) time. It was also hypothesized that these changes in EI and EE would be associated with changes in REM sleep between the control and advanced wake-time sessions.

2. Materials and methods

2.1. Participants

Eighteen participants (12 men and 6 women) completed all sessions. Participants were between the ages of 18–45 years, non-smokers, weight stable (± 4 kg) within the last 6 months, did not have heart problems or diabetes, did not take medication which may affect appetite or sleep, and reported not performing shift work nor taking regular daytime naps. All participants reported having habitual sleep durations of 7–9 h/night. Only women taking monophasic combined estrogen-progesterone birth control were recruited to control for sex-steroid hormone effects on

sleep parameters [17] and EI [18]. This study was conducted according to the guidelines laid down in the Declaration of Helsinki and the University of Ottawa ethics committee approved all procedures involving human participants. Written informed consent was obtained from all participants.

2.2. Design and procedures

Participants took part in a preliminary session followed by 2 weeks of sleep-wake monitoring with accelerometry and sleep diaries, an in-lab habituation night followed by a recovery night at home, and 3 experimental sessions. Fig. 1 presents an overview of the sleep protocol for each experimental session. A washout period of at least 7 days separated each experimental session. Participants were instructed not to consume alcohol or exercise for at least 24 h prior to the preliminary and experimental sessions. They were also asked not to consume caffeinated products after 12 h00 (noon), and to wash their hair in order to facilitate electrode installation on the day of each experimental session. Lastly, participants were asked if they felt well rested at the start of each experimental session. Compliance to these instructions was verified by self-report at the start of each session.

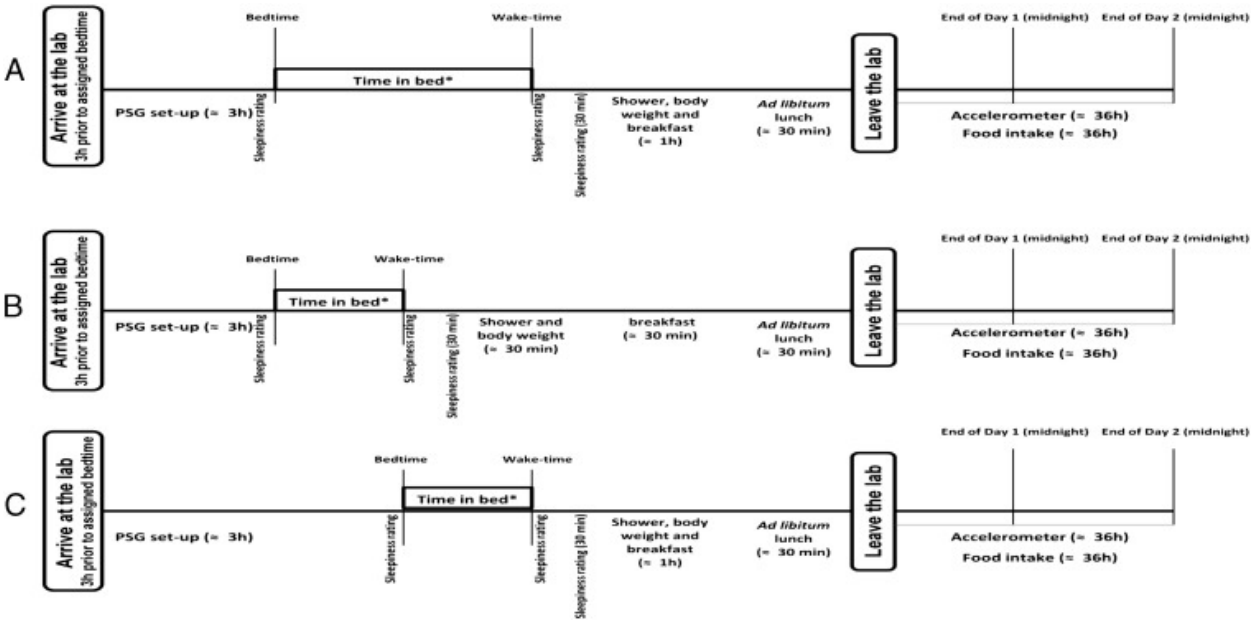

Fig. 1. Overview of the sleep protocol applied during each experimental session.
A) Control session; B) Sleep restriction with an advanced wake-time; C) Sleep restriction with a delayed bedtime
*Based on 2 weeks of accelerometry data.

2.3. Preliminary session

Participants arrived at the lab at 8 h00 following an imposed 12 h overnight fast. At this time, height, body weight and body composition were measured. Participants were then provided with *ad libitum* quantities of the following foods for breakfast: whole-wheat toast (*D'Italiano*®; 4 slices), strawberry jam (*Smuckers*®; 60 g), peanut butter (*Kraft Smooth Peanut Butter*®; 60 g), cheddar cheese (*Cracker Barrel Marble Cheddar Cheese*®; 42 g) and orange juice (*Tropicana*®; 500 g). They were given 15 min to eat as much or as little as they wanted. The measured quantity

and composition of the consumed breakfast was provided to them during each experimental session, and they were instructed to consume the breakfast in its entirety during these sessions. Hence, the energy content and composition of the breakfast varied between participants, but were constant across sessions for the same participant. To assess habitual chronotype and degrees of daytime sleepiness, participants completed the Horne-Ostberg Morningness-Eveningness Questionnaire [19] and the Epworth sleepiness scale [20], respectively. Participants with extreme morning or evening chronotypes (scores ≥ 70 and ≤ 30, respectively) ($n = 0$) and/or frequent feelings of daytime sleepiness (score ≥ 10) ($n = 1$) were excluded from further study participation. Lastly, participants were asked to wear an accelerometer (SenseWear Pro 3 Armbands©, HealthWear Bodymedia, Pittsburgh, PA, USA) and to complete a sleep diary over 2 weeks to assess habitual sleep patterns (bedtime, wake-time, sleep duration and sleep efficiency). Participants were excluded from further participation if they had a mean sleep efficiency [(sleep time/total time in bed) $*$ 100] over 2 weeks < 80% ($n = 1$). These data were also used to tailor the experimental interventions to each participant, and to better capture inter-individual representations of habitual sleep patterns.

2.4. Experimental sessions

All participants took part in 2 habituation nights, which included an in-lab session during which the entire polysomnography (PSG) set-up was performed, followed by a recovery night at home. The first in-lab habituation night was used to exclude participants with sleep disorders (*e.g.* restless leg syndrome, sleep apnea), and to provide an adaptation to the experimental settings used for in-lab sleep assessment. No participants were excluded based on detected sleep disorders, but 1 participant was excluded from further testing because of a very low sleep efficiency during this session (sleep efficiency in this instance was ≈ 20%).

Following this session, participants took part in 3 randomized crossover sessions: control (habitual bed- and wake-times), 50% sleep restriction with a habitual bedtime and advanced wake-time and 50% sleep restriction with a delayed bedtime and habitual wake-time. Each session followed the same protocol and differed only in the amount of total time in bed, and the assigned bed- or wake-time. Since the assigned bed- and wake-times slightly differed between participants (range for bedtime: 22 h16-24 h40, wake-time: 6 h18-8 h37), the time at which each test was administered also differed according to this same range between participants, but remained the same for each participant across sessions. Participants were evenly randomized for the order of experimental sessions (*i.e.* 6 participants started with each of the 3 experimental sessions). Participants arrived at the lab 3 h prior to their set bedtime to allow enough time to place the electrodes (≈ 90 min), set up the polysomnogram (≈ 30 min) and allow for some downtime before bedtime (≈ 60 min). When forced to remain awake during the night and the following morning, participants took part in sedentary activities (*e.g.* reading, watching movies), and remained inside the lab with the researchers. Upon awakening, participants took a shower. Prior to breakfast consumption, which was set at the same time during each session, body weight was measured. Participants also had access to an *ad libitum* lunch, which was consumed inside the lab before being given *ad libitum* quantities of self-selected food items in packed containers for the remainder of the day (day 1; upon leaving the lab until midnight or ≈ 18 h) and the following day (day 2; midnight to midnight or 24 h) [21], along with an accelerometer to wear for this same time period. Lastly, they were asked to bring back the remaining food and all

containers, along with the accelerometer, within the few days following the end of data collection.

2.5. Anthropometric measurements

Standing height was measured, without shoes, to the nearest centimeter using a Tanita HR-100 height rod (Tanita Corporation of America Inc., Arlington Heights, IL, USA). Body weight and body composition (body fat %) were measured using a standard beam scale (HR-100; BWB-800AS, Tanita Corporation, Arlington Heights, IL, USA) and DXA scanner (Lunar Prodigy, General Electric, Madison, WI, USA), respectively. Standing height and body composition were only measured during the preliminary session, whereas body weight was measured prior to breakfast during the 3 experimental sessions.

2.6. Sleep assessment and sleepiness ratings

Sleep was recorded using EEG (C3, C4, O1, O2, F3 and F4), EMG (bipolar submental) and EOG on a Medipalm 22 (Braebon Medical Corporation, Kanata, Ontario, Canada), with the Pursuit Sleep Software (Braebon Medical Corporation, Kanata, Ontario, Canada) inside the lab. Inferior tibialis EMG and naso-oral thermistor signals were also recorded during the in-lab adaptation night. All PSG recordings were scored independently by 2 judges according to the AASM [22] criteria using 30-s epochs; discrepancies were resolved by mutual agreement. The following sleep variables were extracted: total sleep duration in minutes (from sleep onset to wake-time, minus wake after sleep onset), sleep latency (elapsed time between the set bedtime and 10 min of stage 1 or 20 s of any other sleep stage), % of sleep efficiency [(sleep time/time in bed) $*$ 100], wake after sleep onset (WASO) in minutes, as well as the absolute amount of time spent in each sleep stage (stages 1 and 2 sleep, SWS and REM sleep).

Feelings of sleepiness were assessed prior to bedtime, immediately upon awakening and 30 min post-awakening with a 100-mm computerized visual analogue scale [23] with the following question: “How sleepy do you feel?” (Alert/not sleepy at all - very sleepy/sleep onset soon).

2.7. *Ad libitum* energy intake

Ad libitum energy and macronutrient intakes were measured with a validated food menu [21]. This food menu is a list of 62 different items (*e.g.* lasagna, chicken stir-fry, 6 choices of fruits and vegetables, milk, chips, chocolate). Participants were asked to select the food items from this menu that they may want to consume during lunch (consumed inside the lab), the remainder of that day (end of the session to midnight; consumed outside the lab), and the entire following day (midnight to midnight; consumed outside the lab). Two portions of each selected food item was prepared, weighed and served (inside the lab) or packed into separate containers/bottles and placed into a cooler for the participants to bring home with them for the remaining 36 h of measurements (outside the lab). Participants were asked to bring the remaining food items/leftovers, wrappings and containers back to the lab following the end of this measurement period. The remaining items following the in-lab lunch and out-of-lab measurements were weighed, and the energy and macronutrient intakes of the participants for both days across sessions were determined and analyzed with the Food Processor SQL software (version 9.6.2;

ESHA Research). Standard breakfast intake, in-lab lunch and EI for the remainder of the day (end of session to midnight) were combined, and provided energy and macronutrient intake values for day 1. Energy and macronutrient intakes for day 2 were measured from midnight to midnight the following day. One participant did not bring back all food containers at the end of 1 session; hence, the results of 17 participants are presented herein for day 1 and day 2 energy and macronutrient intakes.

2.8. Out-of-lab energy expenditure and activity times

Participants were given a biaxial accelerometer (SenseWear Pro 3 Armbands©, HealthWear Bodymedia, Pittsburgh, PA, USA) prior to leaving the lab during each experimental session, and were instructed to wear the accelerometer at all times, including when sleeping. The accelerometer was placed around the upper arm (mid-distance between the acromion and the olecranon). The SenseWear Professional software (version 7.0, Bodymedia, Pittsburgh, PA, USA) was used to retrieve the data once the accelerometer was returned to the lab. Collected data included: total EE (kilocalories), sedentary time (minutes; < 3 METs), moderate-intensity PA time (minutes; 3–6 METs), vigorous-intensity PA time (minutes; > 6 METs) and estimated sleep time (minutes). Estimated bed- and wake-times, sleep duration and sleep efficiency on night 2 (outside the lab; following PSG and in-lab assessments) were also computed from this device. This device showed overall intraclass correlations above 0.8 when comparing EE with doubly-labeled water [24] and sleep parameters with PSG [25], thus indicating good agreement when assessing these variables with this tool.

2.9. Statistical analyses

Statistical analyses were performed using SPSS (version 17.0; SPSS Inc., Chicago, IL, USA). One-way repeated measures ANOVA tests (for normally distributed data) and the Friedman Exact non-parametric test (for non-normally distributed data according to the Shapiro Wilk test) were used to determine the main effects of sleep condition on body weight, in-lab sleep variables (sleep duration, sleep efficiency and absolute sleep stage durations), energy and macronutrient intakes (days 1 and 2), EE (days 1 and 2), activity time (sleep, waking sedentary, moderate- and vigorous-intensity; days 1 and 2) and sleepiness ratings. Relative sleep stage duration (%) was compared between both sleep restriction conditions with a paired sample *t*-test since the prescribed sleep durations for these sessions were identical. Since significant differences in accelerometer wear time were noted on day 2 (1273 ± 173, 1372 ± 78, 1363 ± 97; $P = 0.01$; partial $\eta^2 = 0.23$), the EE per minute of wear time (kilocalories/min), as well as the relative activity times (%) were compared between sessions. The Wilcoxon Signed Ranks Test was used as a *post-hoc* test to assess potential differences between sessions for variables that were not normally distributed according to the Shapiro-Wilk test. For normally distributed data, *post-hoc* tests with LSD adjustments were used to determine where significant differences existed. Linear regression models were computed to assess the strength of the associations between changes in absolute sleep stage durations with changes in EI and EE on day 1 between sessions. Sex, age and delta sleep duration between the compared sessions were added as covariates to these models. Values are presented as means ± standard deviations. Differences with *P*-values < 0.05 were considered statistically significant.

3. Results

3.1. Participant characteristics, in-lab sleep assessment and sleepiness ratings

Participant characteristics are presented in Table 1. No significant differences in body weight were noted between sessions (69.2 ± 9.2, 69.4 ± 9.3, 69.2 ± 9.4; $P = 0.72$; partial $\eta^2 = 0.02$), a crude indication of energy balance maintenance. Table 2 presents the sleep parameters measured with PSG and accelerometry during each session. Sleep duration was lower during the advanced wake-time *vs.* delayed bedtime condition. Sleep efficiency (overall and after sleep onset) was greater during the delayed bedtime session *vs.* the control and advanced wake-time sessions. Stages 1 and 2 sleep durations were higher, and REM sleep duration was lower, during the advanced wake-time *vs.* delayed bedtime condition. SWS was only higher during the control *vs.* advanced wake-time condition. When comparing relative sleep stage time between both sleep restriction conditions, stages 1 and 2 sleep durations were higher, and REM sleep duration lower, during the advanced wake-time *vs.* delayed bedtime condition. No differences were noted between relative SWS between sleep restriction conditions. Lastly, sleepiness ratings were higher prior to bedtime when it was delayed, and upon awakening in both sleep restriction conditions *vs.* control.

Table 1. Participant characteristics ($n = 18$).

	Mean ± SD	**Range**
Age (years)	23 ± 4	18–33
Body mass index (kg/m^2)	22.7 ± 2.7	19–30
Body fat (%)	18.8 ± 10.1	8–48
Standard breakfast intake (grams)	500 ± 147	257–742
Standard breakfast intake (kcal)	779 ± 240	427–1109
Carbohydrate intake (kcal)	406 ± 121	192–612
Fat intake (kcal)	292 ± 110	136–495
Protein intake (kcal)	109 ± 34	56–170
Total score on the Epworth Sleepiness scales (0 − 10)	6 ± 2	3–9
Total score on the Horne-Ostberg Morning-Eveningness Questionnaire (30–70)	54 ± 8	37–64
Habitual time in bed (min)*	490 ± 38	437–582
Habitual sleep efficiency [(sleep time/total time in bed) * 100] (%)*	86 ± 4	80–91 ($n = 16 < 90\%$; $n = 2 \geq 90\%$)
Habitual sleep efficiency [(sleep time/time in bed after sleep onset and before wake time) * 100] (%)*	91 ± 4	83–96 ($n = 6 < 90\%$; $n = 12 \geq 90\%$)
Habitual bedtime (24-h clock)	23 h27 ± 37 min	22 h17-24 h40
Habitual wake-time (24-h clock)	7 h37 ± 38 min	6 h18-8 h37
Habitual sleep timing midpoint (24-h clock)	3 h32 ± 32 min	2 h40-4 h25

*Based on data collected for 14 days with accelerometry.
Note: kcal, kilocalories; SD, standard deviation.

Table 2. In-lab (polysomnography) and out-of-lab (accelerometry) sleep parameters during each session (n = 18).

	Control	Advanced wake-time	Delayed bedtime	Main effect analysis
	Mean ± SD	**Mean ± SD**	**Mean ± SD**	***P*-value; partial η^{2*}**
In-lab (polysomnography)				
Sleep duration (min)	463 ± 30[a]	229 ± 17[b]	236 ± 17[c]	$P = 0.0001$; partial $\eta^2 = 0.99$
Sleep efficiency [(sleep time/total time in bed) * 100] (%)	95 ± 3[a]	93 ± 4[a]	97 ± 2[b]	$P = 0.001$
Sleep efficiency [(sleep time/time in bed after sleep onset) * 100] (%)	96 ± 2[a]	96 ± 3[a]	98 ± 1[b]	$P = 0.004$
Stage 1 sleep (min)	18 ± 10[a]	7 ± 4[b]	4 ± 3[c]	$P = 0.0001$; partial $\eta^2 = 0.66$
Stage 2 sleep (min)	245 ± 35[a]	113 ± 29[b]	101 ± 31[c]	$P = 0.0001$; partial $\eta^2 = 0.95$
SWS (min)	92 ± 32[a]	76 ± 33[b]	80 ± 31[a]	$P = 0.03$; partial $\eta^2 = 0.20$
REM sleep (min)	108 ± 24[a]	34 ± 7[b]	51 ± 17[c]	$P = 0.0001$; partial $\eta^2 = 0.91$
Stage 1 sleep (%)	NA**	2.9 ± 1.5[a]	1.8 ± 1.0[b]	$P = 0.01$; partial $\eta^2 = 0.36$
Stage 2 sleep (%)	NA**	47.6 ± 12.5[a]	41.9 ± 13.0[b]	$P = 0.02$; partial $\eta^2 = 0.28$
Stage 3 sleep (%)	NA**	31.8 ± 14.2[a]	33.1 ± 12.9[a]	$P = 0.52$; partial $\eta^2 = 0.03$
REM sleep (%)	NA**	14.2 ± 2.9[a]	21.3 ± 7.1[b]	$P = 0.0001$; partial $\eta^2 = 0.59$
Sleepiness ratings (0 − 100)				
Prior to bedtime	65 ± 14[a]	63 ± 22[a]	83 ± 16[b]	$P = 0.001$
Upon awakening	44 ± 26[a]	68 ± 20[b]	60 ± 25[b]	$P = 0.001$
30 min post-awakening	22 ± 16[a]	51 ± 19[b]	45 ± 26[b]	$P = 0.0001$; partial $\eta^2 = 0.46$
Out-of-lab (accelerometry)				
Sleep duration (min)	466 ± 64[a]	512 ± 81[a]	488 ± 131[a]	$P = 0.16$
Sleep efficiency [(sleep time/time in bed) * 100] (%)	87 ± 4[a]	88 ± 6[a]	90 ± 5[a]	$P = 0.16$; partial $\eta^2 = 0.10$
Sleep efficiency [(sleep time/time in bed after sleep onset and before wake time) * 100] (%)	92 ± 4[a]	94 ± 5[a]	94 ± 4[a]	$P = 0.34$; partial $\eta^2 = 0.06$
Bedtime (24-h clock)	23 h23 ± 1h16[a]	22 h40 ± 55min[a]	23 h37 ± 1h38[a]	$P = 0.14$
Wake-time (24-h clock)	7 h57 ± 1h29[a]	8 h09 ± 1h37[a]	8 h19 ± 1h34[a]	$P = 0.70$; partial $\eta^2 = 0.02$

Note: Means not sharing the same letter are significantly different from each other ($P < 0.05$).
*Partial η 2 were not available for variables that were compared using the Friedman Exact non-parametric test.
**It is not possible to compare relative sleep stage time (%) between the control and sleep restriction conditions because of the differences in prescribed time in bed between these conditions.
REM, rapid eye movement; SWS, slow-wave sleep; SD, standard deviation.

3.2. Energy intake and energy expenditure

Table 3 presents data for energy and macronutrient intakes, in addition to relative EE and activity times from accelerometry on days 1 and 2. No differences in energy and macronutrient intakes were noted for day 1. Greater carbohydrate intakes, along with a trend for increased EI, were noted during the delayed bedtime *vs.* control session on day 2. A trend for greater EE on day 1 during the delayed bedtime *vs.* control session was also noted. Vigorous-intensity PA time was higher during the advanced wake-time *vs.* delayed bedtime session on day 1. Moderate-intensity PA time was higher during the delayed bedtime compared to the advanced wake-time and control sessions on day 1. No differences in sedentary and sleep times were noted between sessions for days 1 and 2, nor were there significant differences in sleep parameters assessed with accelerometry outside of the lab.

Table 3. *Ad libitum* energy and macronutrient intakes (n = 17), as well as relative energy expenditure and activity times (n = 18) during each session.

	Control	**Advanced wake-time**	**Delayed bedtime**	**Main effect analysis**
	Mean ± SD	**Mean ± SD**	**Mean ± SD**	**P value; partial η^2**
Total energy intake - Day 1 (kcal)	2686 ± 765[a]	2768 ± 825[a]	2844 ± 735[a]	P = 0.42; partial η^2 = 0.05
Carbohydrate intake (*kcal*)	1507 ± 442 [a]	1544 ± 496 [a]	1623 ± 483 [a]	P = 0.33; partial η^2 = 0.07
Fat intake (*kcal*)	854 ± 304 [a]	874 ± 313 [a]	870 ± 238 [a]	P = 0.92; partial η^2 = 0.01
Protein intake (*kcal*)	385 ± 139 [a]	401 ± 130 [a]	411 ± 126 [a]	P = 0.32; partial η^2 = 0.07
Total energy expenditure - Day 1 (kcal/min)	2.2 ± 0.6 [a]	2.3 ± 0.5 [a]	2.5 ± 0.9 [a]	P = 0.05; partial η^2 = 0.16
Vigorous-intensity time (%)	1.6 ± 2.3[a, b]	2.7 ± 3.0[a]	1.3 ± 2.4[b]	P = 0.004
Moderate-intensity time (%)	16.1 ± 10.6[a]	17.5 ± 11.8[a, b]	26.6 ± 19.9[b]	P = 0.01; partial η^2 = 0.24
Sedentary time (%)	73.0 ± 13.1 [a]	66.4 ± 16.9 [a]	62.3 ± 18.4 [a]	P = 0.06; partial η^2 = 0.07
Sleep time (%)	9.3 ± 6.2 [a]	13.4 ± 10.9 [a]	9.8 ± 11.7 [a]	P = 0.61
Total energy intake - Day 2 (kcal)	2345 ± 816[a]	2534 ± 783[a]	2658 ± 899[a]	P = 0.05; partial η^2 = 0.17
Carbohydrate intake (*kcal*)	1386 ± 513[a]	1453 ± 440[a]	1579 ± 571[b]	P = 0.04; partial η^2 = 0.18
Fat intake (*kcal*)	649 ± 291[a]	755 ± 316[a]	743 ± 281[a]	P = 0.10; partial η^2 = 0.13
Protein intake (*kcal*)	354 ± 138[a]	377 ± 142[a]	385 ± 145[a]	P = 0.44; partial η^2 = 0.05
Total energy expenditure – Day 2 (kcal/min)	2.0 ± 0.5 [a]	2.1 ± 0.3 [a]	2.2 ± 0.6 [a]	P = 0.23
Vigorous-intensity time (%)	0.9 ± 1.2[a]	1.1 ± 2.1[a]	1.1 ± 1.5[a]	P = 0.14
Moderate-intensity time (%)	12.6 ± 9.3[a]	12.6 ± 7.3[a]	16.6 ± 10.0[a]	P = 0.53
Waking sedentary time (%)	52.3 ± 6.0[a]	51.6 ± 9.5[a]	48.6 ± 13.1[a]	P = 0.45; partial η^2 = 0.05
Sleep time (%)	35.0 ± 7.8[a]	34.0 ± 7.1[a]	33.7 ± 8.7[a]	P = 0.99

Note: Means not sharing the same letter are significantly different from each other (P < 0.05).
kcal, kilocalories; SD, standard deviation.

3.3. Linear regression model results

Greater stage 1 sleep duration was associated with greater day 1 EI between both sleep restriction conditions in the adjusted linear regression model (β = 110 kcal, 95% CI for β = 42 to 177 kcal; P = 0.004). A trend was also noted for increased EI on day 1 with lower REM sleep duration between both sleep restriction conditions (β = − 20 kcal, 95% CI for β = − 41 to 2 kcal; P = 0.07). Identical results were noted when assessing the strength of the associations between changes in relative (%) stage 1 and REM sleep durations with EI between sleep restriction conditions on day 1. No other significant associations were noted between changes in absolute sleep stage time and changes in energy balance parameters (results not shown).

4. Discussion

To our knowledge, this is the first study to examine the effects of an imposed sleep restriction combined with altered bed- or wake-times on objective measures of EI and EE with a randomized crossover design. This study also used 2 weeks of accelerometry data for each participant to personalize the bed- and wake-times for the experimental sessions, which offers optimal control over inter-individual variations in circadian rhythms. This may in part explain the relatively high in-lab sleep efficiency values observed in this study. Collectively, our results suggest that carbohydrate intake on day 2 was greater following sleep restriction with a delayed bedtime compared to the control session. Additionally, moderate-intensity PA time was greater on day 1 during the delayed bedtime *vs.* advanced wake-time and control sessions, whereas vigorous-intensity PA time was greater on day 1 following sleep restriction with an advanced wake-time *vs.* sleep restriction with a delayed bedtime. These results do not support our initial hypothesis. The changes in EI and EE between the control and advanced wake-time sessions were not associated with changes in REM sleep duration, thus refuting our second hypothesis. However, when comparing both sleep restriction conditions, greater stage 1 sleep, in addition to a trend for lower REM sleep, durations were associated with greater EI the next day (day 1). This is the first study to report associations between changes in sleep architecture with next day EI and EE between sleep restriction conditions.

Many experimental studies reported greater 24 h EI following sleep restriction *vs.* control sessions [6], [7], [8], [9], [10], [11]. Studies also reported increased intakes of all [10] or specific macronutrients, such as fats [11] or carbohydrates [9], following partial sleep restriction *vs.* habitual sleep duration. Although sleep timing was not evaluated in these studies, the majority employed longer-term (≈ 2–14 nights) sleep restriction conditions, which may explain the greater differences in EI between conditions. A study conducted by Brondel et al. [7] also imposed 1 night of partial sleep restriction, compared to 1 night of habitual sleep duration, and reported greater EI following partial sleep restriction. The present study did not observe a significant difference in EI between sessions on day 1, but did note greater carbohydrate intake on day 2 following partial sleep restriction with a delayed bedtime. Although a mere hypothesis, it is possible that the environment in which EI was measured (in-lab *vs.* outside the lab), and some of the restrictions imposed with regards to meal intake inside the lab (standardized breakfast and fixed meal times inside the lab) may have decreased the number of opportunities for spontaneous or increased EI on day 1. Similarly, the present study did not permit EI during the time forced to remain awake during the sleep restriction sessions. If *ad libitum* EI would have been permitted during these overnight hours, EI would have most likely been higher. Indeed, greater 24 h EI following imposed sleep restriction in previous studies were often characterized by greater late night and/or post-dinner snack intake during the sleep restriction condition [9], [10], [12]. Hence, the timing of EI under free-living conditions should be monitored in future studies, as this may help further explain the sleep-EI link [26].

Relative moderate-intensity PA time was greater during the delayed bedtime *vs.* advanced wake-time and control sessions, whereas relative vigorous-intensity PA time was higher following sleep restriction with an advanced wake-time *vs.* delayed bedtime. The only other study to note greater PA participation following imposed partial sleep restriction [7] reported that this increase in PA participation occurred when no restrictions in PA choices were imposed on participants. In

the present study, PA participation was only measured when participants left the lab and no limitations on their PA choices were imposed. However, they were instructed not to go to bed until their habitual bedtime on the evening following each sleep manipulation. It is possible that these participants resorted to PA following sleep restriction to combat feelings of fatigue during the day that immediately followed the sleep manipulations (day 1). A meta-analysis by Puetz [27] revealed that PA participation was associated with a reduced risk of experiencing feelings of low energy and fatigue in habitually active *vs.* sedentary adults. Considering that this study's sample included mostly active individuals (*i.e.* ≈ 15–23% of total time spent performing moderate-vigorous intensity PA), resorting to PA participation to alleviate feelings of fatigue and maintain wakefulness until their habitual bedtime may be seen as a viable solution for these participants. Studies comparing energy balance measurements in individuals with habitually high- *vs.* low-levels of PA participation following sleep restriction with altered sleep timing are needed to investigate this hypothesis.

The linear regression models conducted revealed that greater stage 1 sleep, in addition to a trend for lower REM sleep, durations were associated with greater EI the next day (day 1) between both sleep restriction conditions. A study by Shechter et al. [15] reported significant inverse associations between relative stage 2, SWS and REM sleep durations with EI between a habitual and partial sleep restriction condition [15]. Although no cause-and-effect associations can be drawn from the present findings, it is possible that participants with greater amounts of restorative sleep (*i.e.* less stage 1 and more REM sleep) may be able to exert greater control over food intake, even though total sleep time is reduced by 50%. These findings also suggest that inter-individual differences in sleep quality in response to imposed sleep restriction may impact EI differently the next day. Further characterization of individuals by evaluating personality traits, morning-evening preference [28] and/or differences in cognitive inhibition [29] following sleep restriction are needed, as these may be strong moderators of changes in EI following sleep restriction.

The present findings are limited to a small sample size of healthy, physically active adults with habitually high sleep efficiencies. This limits generalizability to other populations, including individuals with sleep complaints or sleep disorders. Biaxial accelerometry provides an estimation of sleep-wake activities. The use of doubly-labeled water in conjunction with a triaxial accelerometer and PSG on the 2nd night would have provided more precise measurements of sleep and waking activities, including physical activity participation. The assessment of sleep with PSG and all outcome variables were only conducted for 1 night and 36 h post-intervention in each condition, which does not account for day-to-day variations, and limits the comparison of results with studies imposing prolonged sleep restrictions. Lastly, PA and EI were not permitted during the time that participants were forced to remain awake inside the lab, which may have attenuated some of the effects of sleep restriction on EI and EE. Future studies with similar objectives and designed to allow for *ad libitum* access to food and exercise during the overnight hours/time forced to remain awake are needed to add to these results.

In conclusion, sleep restriction with a delayed bedtime led to greater carbohydrate intake, whereas relative moderate-vigorous PA time was greater following both sleep restriction sessions. Future studies are needed to further investigate sleep architecture and energy balance parameters in individuals with sleep disorders (*e.g.* insomnia, sleep apnea), as individuals with

sustained poor sleep quality and/or reduced sleep duration may be at an increased risk of weight gain over time [30]. This information would be invaluable given the increasing prevalence of individuals experiencing regular circadian misalignment as a result of shift work, in addition to an increase in the incidences of sleep disorders and voluntary sleep restriction [31].

Conflict of interest disclosure

The authors declare no conflict of interest.

Author contributorship

JM, ÉD and GF formulated the research questions and designed the study. JM, J-FB, LJH, IC, ÉL, RM and AR carried out the experiment. JM, ÉD, J-FB and GF analyzed the data. All authors were involved in writing the paper and had final approval of the submitted and published version.

Acknowledgements

The authors would like to thank the participants for their involvement in this study. J McNeil was a recipient of the Ontario Graduate Scholarship during the time of data collection.

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